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A CLINICAL CONSIDERATION

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SIXTY CASES OF CEREBRAL PARALYSIS IN CHILDREN.

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A CLINICAL CONSIDERATION OF SIXTY CASES OF CEREBRAL PARALYSIS IN CHILDREN.[1]

BY ROBERT W. LOVETT, M.D., BOSTON.

In some 2200 cases of disease in children observed and treated at the Surgical Out-Patient Department of the Children's Hospital, in the four years ending last January, there presented themselves among the cases of motor disturbance, some 60 patients where there seemed reason to believe that the disability was of cerebral origin. Cases of anterior poliomyelitis, or common infantile paralysis outnumbered the cerebral cases three to one at least, but these cases of infantile paralysis are left out of consideration here.

It has been possible to obtain a recent examination and a careful history of the great majority of these 60 cases, the practical out-come of which it is desired very briefly to present, not from the standpoint of the neurologist, but considering simply the gross clinical characteristics of these cases as they were observed. And first, they must be grouped, and they fell naturally enough as a rule, into one of three classes. Hemiplegia, Spastic Paralysis of both legs, and a class of non-descript cases, perhaps best called Incoördination or Idiocy. But this arrangement follows no authority, and represents merely an arbitrary clinical division for convenience in presenting them. Unfortunate, as

[1] Read, and patients exhibited, before the Section for Clinical Medicine, Pathology and Hygiene of the Suffolk District Medical Society, April 11, 1888.

the statistical method is, it seems the only available way of handling so large a group of cases.

Among these 60 cases there were 26 cases of Hemiplegia, half of the right side and half of the left, and about equally divided between the two sexes, and most of the children at the time of observation were between 4 and 8 years old (although they range from a year and a half to 14).

Two-thirds of these cases were hemiplegia of practically the adult type of distribution ; while the remainder represented a combination of hemiplegia, and beginning or advanced spastic paralysis of both legs as well, the significance of which must be considered later.

There was in every case of hemiplegia at the time of observation a certain amount of impairment of motion of the diseased side, with more or less muscular atrophy and bone shortening in some cases where the paralysis had existed for some years. One case showed a shortening of 2 inches in the length of the arm after a paralysis of seven years standing. Three other cases of 4, 7 and 8 years standing showed an inch shortening in the leg or arm. The muscular atrophy, was as a rule, slight and not at all comparable in severity to the muscular atrophy of Infantile Spinal Paralysis of the same duration ; it was ordinarily a difference of less than an inch in the circumference of the arm or leg, but in some cases the limb had wasted to almost nothing but skin and bone. The arm and hand were useless on account of atrophy or ataxia, in only 7 of these cases, while in 14 they were fairly useful, and 5 were not noted. The arm was in all cases affected more severely than the leg, and recovered more slowly. As to the use of the legs, 5 children walked perfectly well, 4 rather poorly, 7 with a bad limp, 7 not at all.

It is interesting to note that where the parents were careful people and had kept the affected limb well rubbed and bent, the limbs were always in a condition of at least partial usefulness. Facial paralysis had been present in at least half the cases and strabismus was present at the time of observation in more than half, adding to the already stupid look of the patients.

Athetosis was noted in only two cases, an unusually small proportion.

The prevalence of mental impairment in these cases of hemiplegia is most striking, and probably exceptionally large; at the same time, the series represents every case of hemiplegia that was seen, however slight the affection. Of these 26 cases there were only 6 that could be accounted of average intelligence, under a most charitable interpretation, and of these one was aphasic and another stuttered most markedly. All the 6 could read. Seven were idiotic; that is, they were unable to talk or care for themselves or understand very much of what was said to them, and their expression was characteristic of the low grade idiot; 8 might perhaps be classed as feebleminded. All but two could talk imperfectly, but they were all easily excitable, passionate and violent. They could not seem to learn and would not mind what was said. Their faces had a stupid or epileptic look, and all but one were subject to convulsions. Four were of a still higher grade and were presentable, but by no means up to the average, 1 could go to school, but 2 of the others, five years old, did not know their letters: they might be classed as backward; and 1 was one and a half years old, too young to be classed.

Of the 26, 7 were aphasic, and four times this was with left-sided hemiplegia and three times with right-sided. When larger groups of cases are considered

right-sided hemiplegia shows 47 per cent. of accompanying aphasia while left-sided only 26 per cent. I quote from 160 cases analysed by Wallenburg.[2]

The first point of interest in the discussion of the etiology of these cases lies in the question of whether they were born after a hard or an easy labor. Mr. Little of London was the first to call attention to the associations of difficult labor and spastic paralysis, and in 1862[3] appeared his paper which has become classical, "On the Influence of Abnormal Parturition, etc., upon the future mental and physical condition of the child," in which he tabulated some 63 cases of spastic paralysis and hemiplegia, in all of which he found that it was due to difficult labor. Ross[4] twenty years later elaborated the point somewhat more fully, and quoted authorities to show the liability of the new-born to meningeal hæmorrhage. Weber[4] made 161 autopsies of new-born children, and in 81 cases where the spinal canal and head were opened, 33 times there was extravasation of blood from the spinal and cerebral meninges. Of 64 cases of foot extractions examined by Ruge[4] there was rupture of the vertebral column in 8.

There was no such universal difficulty of labor in these 26 cases of hemiplegia, there were 5 where it were not possible to obtain a reliable account of the labor. In the remaining 21 cases, 8 children were born by an easy, normal labor, on the testimony of the mother. In 4 cases the labor was described by the mother as "very hard." In 7 cases instruments were used; in the 7 instrumental cases, once version was done and once the child was born asphyxiated, and once prematurely at 7 months. Two of the children have, at

[2] Jahrbuch f. Klide. 1886. N. F., xxiv, p. 384.
[3] Obstet. Trans. London, 1862.
[4] Brain, Vol. 344.

the age of 4 and 8 years a depression in the skull, on the side opposite to the paralysis, left from the use of the forceps. In the kindred class of spastic paralysis considered here for comparison, of 12 cases, 9 were born by an easy and normal labor, 1 was a breech presentation, and 2 were born before term at 8 months. Since then 17 of these 33 cases of cerebral paralysis (more than 50 per cent.) were born by normal labor which the mothers described as easy; it seems reasonable to conclude that the influence of difficult labor in producing cerebral paralysis must have been very much overestimated. Probably, accounts of the labors from unprejudiced persons would show a much higher percentage of normal labors. In speaking of the class of feeble-minded children in general, Dr. Langdon Down [5] says: "I found that among the great number of feeble-minded children about whom I could get thoroughly reliable accounts, in only 3 per cent. had the forceps or any other instrument been used," and he also quotes the late Dr. Ramsbotham as telling him "that the cases were very few in which he could trace any cerebral lesion as resulting from the employment of the forceps."

Of these 26 hemiplegia cases, 7 were noted immediately after birth, 5 cases in the first year, 12 cases in the second year, and over two years only two cases, both at the age of five years; once after an attack of diphtheria, and once it developed without warning or sickness of any sort. It is interesting to note how evenly distributed the liability to cerebral paralysis is for the first six or seven years of life.

Of Wallenburg's 160 cases, 19 showed paralysis at birth or immediately after, in the first year, 35, second year, 29, third year, 17, fourth year, 9, fifth year, 9, sixth year, 3, seventh year, 6, eighth year, 3. Some 150

[5] Mental affections of childhood and youth. London, 1887, p. 44.

cases of anterior polio-myelitis, however, considered by Sinkler,[6] showed a different distribution, and were sharply limited to the first three years of life, six-sevenths of all cases occurring in that time. The number of cases of cerebral paralysis increases at the time of the second dentition.

An illness of some sort marked the onset of the disease almost always; sometimes only a severe crying spell, or what was considered bowel irritation, or indigestion, often convulsions, ushered in the paralysis; having been present in 12 of the 26 cases. This is not far from the usual proportion; but only eight times did they mark the beginning of the paralysis; once they preceded the paralysis by one year, and three times they followed its establishment at intervals of from four months to five years. But as only one of the children under consideration has reached puberty, it is hardly time perhaps to expect the full development of the epileptic convulsions said to appear at that time. Cases are recorded where twelve and thirty years have elapsed before the appearance of convulsions.[7] In only one case could I associate the beginning of the paralysis with any acute infectious disease, and that was the diphtheria noted above, but the exanthemata are often the cause, no less than 28 cases having been reported as due to scarlet fever, and measles, and typhoid, mumps, whooping cough, and even vaccination are accountable for 11 more reported cases.[8] The pathology of these hemiplegic cases can best be considered after speaking of the cases of pure spastic paralysis.

[6] Sinkler. Am. Jour. Med. Sciences, April, 1885.
[7] Gowers. Epilepsy, London, 1880.
[8] Wallenburg and Gowers. Loc cit.
Marie. Prog. Med., No. 36.
Richardiere. Etude sur les scleroses enceph. priere. de l'unfant, Gliene de Paris.
Jeudeassik and Marie. Auk de Phys. non et Potte, V. 51, 1885.

"Spastic paralysis" is perhaps the commonest name for the condition also known as "Little's disease," "tetanoid pseudo paraplegia,"[9] "spastic spinal paralysis,"[10] "permanent tetanus of the extremities,"[11] and so on. It is only of late years that the affection has been clearly recognized, and since the work of Charcot and Erb it has, of course, been known to be associated with degeneration of the lateral columns of the cord. Very briefly, the condition is characterized by a persistent stiffness and constant spasm of the muscles of the legs and sometimes of the arms; the legs are straight and rigid, and the feet are extended, and when an attempt is made to walk the child stands on tiptoe, and often the adductor spasm is so great that the legs are crossed. There is no marked wasting of the muscles, and the deep reflexes are much increased. The walk is almost characteristic, a clinging gait, in which the feet are scraped along the floor with much effort and straining at every step, if indeed the spasm is not so great that walking at all is out of the question.

There were 16 cases of this bilateral spastic paralysis in the 60 cases, not counting the 7 cases where it was combined with hemiplegia; and the age of the patients ranged from two to twelve years at the time of observation. In 4 cases the notes were imperfect, and the paralysis was noted immediately after birth in eight of the remaining twelve, but in only three was it associated with abnormality of labor, as we have already seen. In the other 5 cases where it was noted at birth no more definite causes could be assigned than a fall during pregnancy, worry during pregnancy, except in one case where the child had a long

[9] Seguin. Opera minora.
[10] Erb and Seeligmuller. Jahrbuch. f. Klide, N.F. xiii, 1879, 226 and 315.
[11] Stoomyer. Hdbch du Chir., 1867, 17 and 174.

and severe crying spell immediately after birth, which of course suggests the occurrence of meningeal hæmorrhage. The family history in all these cases was good.

Of the four cases in which paralysis was not noted immediately after birth, one was clearly traumatic, coming on in a girl eighteen months old, after a fall of some thirty-six feet; and in another case it gradually developed about the age of six years. The other two children were brother and sister, and the affection developed after an acute sickness called by the attendant "slow fever," which they both had at about the same time, when one was two years old and the other only a few months, but the fact that one of the children showed congenital absence of the uvula does not lend much strength to the parents' theory. One of the congenital cases was very likely syphilitic in origin, as the father had the disease at the time of the child's birth, but the notes were not definite on the point.

Here the mental impairment was even more general than in hemiplegia, for no one of the 12 cases where the records are complete could be classed as intelligent. 6 were low grade idiots, 3 were very stupid, but could understand nearly all that was said to them, two were backward and mischievous, while one appeared almost bright, but was said by his mother to have "no sense;" 7 were completely aphasic, while 5 could talk more or less.

In 9 of the 12 there was a strabismus, and in every case a stupid or idiotic countenance. Of the 16 cases 12 were unable to walk at all, either from the persistent muscular rigidity of the legs, or from the mental deficiency also. Two could walk alone, one with a chair and one with crutches. Of the 16 cases, in 5 the cross-legged deformity was noted, the legs being crossed by the adductor spasm on trying to walk; pro-

gression in this method is, of course, impossible. In 7 cases the hands were noted as also impaired, and one child was said by the mother to have taken hold of things with crossed hands; that is, he used his left hand to take hold of things on his right, and the right hand for things on his left, but that, of course, is very doubtful.

The patella reflexes were in all cases much increased when the muscles were not too rigid to prevent it from being seen, and the muscles offered always a tetanic resistance to attempted manipulation. When the rigidity of the legs was not too great, ankle-clonus was also present, but this was found in proportionately few cases.

There were certain vices of malformation — the uvula was absent in one case, and in one there was a co-existent inguinal hernia, a high arch to the roof of the mouth was present in a certain number of both these and the hæmiplegic cases, but in by no means so large a proportion as Down found it, who analyzed 200 cases, and found an abnormally high palate in 41%. The children were much given to drooling, and one case of this sort was sent to Dr. F. H. Hooper, who found an adenoid growth in the nasopharynx, and removed it, with much relief to the patient; and one case of hemiplegia, with beginning spastic symptoms, had the same trouble, and was relieved in the same way, both with some improvement of the mental condition.

The question of whether spastic paralysis in children is a spinal or cerebral affection has always excited much discussion. Little thought it in many cases spinal, Charcot and Erb in their description of primary lateral sclerosis in the adult, seemed still further to confirm this view although Erb [12] expressly avoided

[12] Erb. Memorabilien aus au Praxis Heilbroun, 1887. 12 Heft.

committing himself to any such views, and suggested a lack of development in the nervous centres as the cause. As Catsaras[13] puts it, "Is the cord-lesion primary, or is it secondary and descending, due to a bilateral cerebral lesion? Recent opinion is perhaps well formulated by Ross,[14] who says, "The opinion that a large number, if not all cases of the spastic paraplegiæ of infancy are caused by porencephalous defect of the motor centres, along with an arrest of development of the corresponding parts of the pyramidal tract, is here advanced with due reserve." Spastic spinal paralysis, then becomes a "double spastic hemiplegia" as McNutt[15] called it, and the existence of primary spinal spastic paralysis becomes a question of doubt.

In this series of cases there was no patient who was free from cerebral symptoms, no case of pure spinal spastic paralysis. One case seemed to be spinal in origin and to have but little cerebral trouble beyond a certain backwardness, and he was sent to Dr. J. J. Putnam for diagnosis, who wrote back that it seemed to him to be of cerebral origin, and he suspected as the cause of it a meningeal hæmorrhage soon after birth, when the child had a violent crying spell which lasted for some time. He added that he thought there was undoubtedly some general cerebral atrophy beside the affection of the motor tract.

Naef,[16] who wrote a very exhaustive paper upon the subject, thought that there was not more than one case of pure spinal spastic paralysis in 1,000 children sick with various ailments, and admitted that the existence of primarily spinal spastic paralysis in the

[13] Catsaras. Ann. Med. Psychol. July, 1887.
[14] Ross. Loc. Cit.
[15] McNutt. Am. Jour. Med. Sci., Apr., 1888. 9, 58.
[16] Naef. Die spast. spin. Paralyse in Kindesalter Inaug. Diss., Zurich, 1885.

child, lacked anatomical proof, and Ross, although he thinks that spinal spastic paralysis exists in children, speaks of its rarity.

All that can be said is that in 2,200 cases of surgical disease, representing really a much larger number of sick children in general, if one is to reckon by that method, no case of spastic spinal paralysis has presented itself.

Before speaking of the great amount of theorizing as to the pathological process causing hemiplegia and spastic paralysis in children, it will be best to see what the recorded autopsies show as the pathological appearances, and I am able to give a summarized account of 77 autopsies upon cases of this sort.[17] There were among these cases 37 times a right hemiplegia; 29 times a left hemiplegia; 3 times a single hemiplegia, the side not noted; 8 times paralysis of both sides (spastic paralysis or double hemiplegia).

From the nature of the disease, autopsies, when the disease is of short standing, are very rare. Most of these recorded, are months or years afterward. Of 12 recent cases where the autopsy occurred within a few weeks or months after the onset of the paralysis, 7 cases showed clearly embolism of the artery of the fissure of Sylvius, 45 showed a hæmorrhage in the hemispheres or ventricle of practically the adult type, and microscopic examination could show no difference between the local condition found there, and in adult hemiplegia; one of these cases (that of Reimer[18]) was examined within six days of the onset of the paralysis. Apart from these 12 cases the others are all of longer standing than a year, most of them as

[17] Wallenburg and Mr. Worth. Loc. Cit.
Kast. Arch. f. Psych., 1887. xviii, 280.
Bullard. Boston Med. and Surg. Jour., Feb. 16, 1888.
Otto, Arch. f. Psych., etc., xvi, 215.
[18] Reimer. Jahrb. f. Kline. W. F. ii, 1877, p. 70.

many as 40 or 50 years after the beginning of the paralysis and showed changes much less definite; but 13 of the more recent cases showed very clearly a local lesion like a cyst, or apoplectic scar, an atrophy and sclerosis clearly localized in the motor region, and from this condition to a general atrophy of a whole hemisphere, every graduation was represented. They all show, with a few exceptions, a gross defect of tissue in the cortex near the fissure of Rolando. It used to be called atrophy of the hemisphere and as such the older cases stand recorded. In some of these cases the meninges showed signs of having been involved as well as the brain, but in most of them it is impossible to say whether the process began in the brain or its membranes. Sixty-three of the cases showed this local or general destruction or non-development of tissue in the motor region. Heschl [19] gave it the name porencephalus, and Kundrat [20] wrote a most extensive paper about it, and now it is known as porencephalon or porencephalous defect, which, as I understand it, means only a lack of tissue in the brain substance from any cause. The name originally arose from the fact that the convolutions converge toward these pits at the bottom of which there is apt to be a communication with the lateral ventricle.

Two cases remain out of the 77 to be accounted for; one [21] showed a diffuse sclerosis of the cortex due to a diffuse, chronic encephalitis and the last (the case of Seeligmüller) [22] was a case of brain tuberculosis with several foci distributed through the brain; where the paralysis is of both legs, sometimes the brain lesion is unlateral and sometimes it involves both sides, as in the well-known case of McNutt.

[19] Prag. Viertelghrsch, lxi, 1859, p. 59
[20] Kundrat. Die Porencephalia Gray, 1882, p. 45.
[21] Kast. Loc. cit.
[22] Seeligmüller. Jahrb. f. Klin. F.W., xiii, p. 760.

This is very briefly all that is known of the pathology of the condition; a lesion of the motor tract, atrophy and retarded development of the brain, and descending degeneration of the pyramidal tracts and lateral columns of the cord; and from the extensive atrophy found in young children at autopsy, it seems that unquestionably sometimes the disease originates in defective development of the nervous centres, especially the pyramidal tracts. Flechsig[23] found these absent in some anencephalic children and in one of Kundrat's cases they were imperfectly developed.

Having then seen that hemiplegia and spastic paralysis differ but little in the original lesion causing the paralysis, it is not surprising that 9 of the twenty-six hemiplegia cases here reported showed spastic paralysis of both legs as well; and the interest of the matter comes in the question of whether hemiplegia in children is apt to go on to spastic paraplegia later, a matter of the greatest practical importance, of course, in the prognosis. In the 17 cases where hemiplegia alone existed, the average duration of the paralysis at the time of observation had been 3.8 years; but in the 9 cases where spastic paralysis was also present the average duration had been 5.9 years, or more than two years longer. It would seem from this that spastic paralysis co-existed with hemiplegia in cases of longer standing than when hemiplegia was found alone. Moreover, what appear to be transition cases from simple hemiplegia to severe spastic paralysis were present. Two of the children, aged 4 and 14 respectively, had hemiplegia which presented the usual signs, but they had also exaggerated tendon reflexes, they walked with something of the clinging gait that distinguishes spastic paralysis, and they were unsteady on their feet, and one of them has

[23] Flechsig. Leitungbalm in Geturee and Rushemark, p. 198.

lately become worse. Possibly they will never go on to any more serious condition than this, but the other 7 cases present fully marked signs of spastic paralysis with those of hemiplegia.

The co-existence of spastic paraplegia and hemiplegia in the adult has not escaped attention. Hadden [24] has written about it, and accounts for its occurrence by the presence of an unusual decussation of the pyramidal tract fibres. Charcot, on the other hand, thinks it due to a second decussation of the motor fibres in the dorsal region.

With regard to the theory of Strümpell, [25] that a large proportion of cases of cerebral paralysis in children are due to a primary local inflammation of the brain-cortex cells, analogous to the inflammation of the cells in the anterior cornua of the cord in infantile paralysis, it can only be said that the theory, however plausible, lacks anatomical proof; even Kast, its latest advocate, admits that. Unless the recent case of Ruhemann be accepted as poliencephalitis, there is nothing resembling a primary local encephalitis in any of the autopsies made early in the disease, and the atrophy found in the later ones is perfectly well explained by the occurrence of such well known processes as embolism, hæmorrhage, thrombosis, as Gowers [26] suggests, and defective development. Ruhemann [27] recently published the autopsy in the case of a child eleven years old, who died in the midst of convulsions. Death came on six hours after the beginning of convulsions, and examination showed in the gray substance near the fissure of Rolando on the right side a small spot of a more translucent color than the

[24] Hadden. St. Thomas Hosp. Rep., 1882, 61.
Pitres. Arch. de Neurol, No. 10.
[25] Strümpell. Jabrbuch f. Klin., N. F., xxii, 1874, 173.
[26] Diseases of the Brain, London.
[27] Cent. f. Kl. Med., 1887, 18.

rest. No microscopical examination was made. No other lesions were present.

With regard to the diagnosis between infantile and spinal paralysis, there is ordinarily little likelihood of confusing the two in the later stages. At the beginning, unless facial paralysis or distinctly hemiplegic convulsions are present to show a cerebral origin, it is practically impossible. Cerebral paralysis ordinarily begins with a convulsion or illness, its distribution is oftenest hemiplegic, which is very rare in infantile paralysis and the tendon reflexes are increased on one or both sides. Wasting of the limb is slight for some time, but ultimately bone-shortening may come on, *and, most important of all, the paralyzed muscles show no qualitative change in their reaction to the galvanic current.* Infantile paralysis begins oftenest, also, with a convulsion or illness. It affects oftenest one limb. Facial paralysis is never present. Wasting begins at once and is very rapid, and the limb is cold and blue. The reflexes are diminished and ultimately lost. *The paralyzed muscles show to the galvanic current the qualitative changes known as the reaction of degeneration.*

The prognosis of these cases of hemiplegia is not very brilliant; the chances are that mental enfeeblement will come on, and possibly spastic paralysis. With good care the paralyzed side can probably be kept from extreme atrophy and those contractions that render the limb useless, and after the shock, nearly all the cases here analyzed improved somewhat in the use of the limb. Epilepsy is very apt to develop at puberty or before.

In spastic paralysis it is safe to assert that the child will probably ultimately be able to walk somewhat if the case is not very severe, and it will probably learn to talk at the age of five or six or seven. Almost all

of the cases here recorded have improved in the last three or four years. There is, of course, no outlook of recovery, but their tendency has been to walk better, to talk a little more clearly and in every way to improve. Two of the forty-two cases have died in the last three years, both apparently of meningitis.

The treatment of these cases can be discussed in very few words, in view of the character of the brain lesion. Inasmuch as it is a defect of tissue, but little is to be expected from operative interference, for there is no pressure to be relieved, nothing to be removed, unless at the time of the occurrence of the paralysis it might be possible to open the skull and turn out a meningeal clot, as McNutt suggests, if any one felt disposed to undertake so great a risk. After paralysis has occurred, of course the harm has been done, and the aim should be to keep the muscles from atrophy, and in as good condition as possible by massage and manipulation, and most important of all, the use of the faradic current to the affected muscles. Galvanization of the head has been proposed, but is denounced by such men as Gaudard [28] and Bernhardt.[29] The child should receive, it is said, not more than four or five applications of the faradic current each week, and each sitting should be from five to fifteen minutes at the most. The mental training forms a most important part of the treatment, but that must be passed over here. No form of apparatus can, of course, benefit the children, unless the heels are so persistently drawn up by the tendo-Achillis that walking is impeded in which case it is easy to do a tenotomy, and, bringing the foot down, retain it by a club-foot shoe. One of the cases of spastic paralysis had received some little benefit in this way.

[28] E. Gaudard. Cont. a l'étude de l'hemipl. cer. inf. These, Geneva, 1881.

[29] Bernhardt. Virch. Arch. Bd. 102, Heft 1, S. 26.

It will be necessary to pass over very briefly the last of the three classes into which the cases were divided. It represents at best a miscellaneous collection which were classed as incoördination or idiocy. Their only excuse for appearing here is the very close outward resemblance that they present on superficial examination to the hemiplegic and spastic cases already considered; but definite paralysis and spastic rigidity of the muscles are absent, and idiocy obscures everything. If one sees them sitting down, the stupid cross-eyed look, the drooping head, the drooling and all, are exactly what one sees in the severe mental enfeeblement of spastic paralysis of hemiplegia, but put the child on his feet and the difference is at once evident. Either he is so limp that he will be unable to bear his weight at all, or he will stand holding his parent's hands with his feet wide apart, his knees bent, and his trunk leaning forward. The whole body sways to and fro with an oscillating movement, and the sense of equilibrium seems almost wanting; if he is let alone, he walks in a staggering uncertain way, with many falls. This describes the state of the best of the 13 children of this sort that I saw, and from this the condition grades off to a disability so great that the child cannot even sit up; when it is propped up the head lops on to one shoulder, the vertebral column fails to support the trunk and bends to a marked degree, and every muscle seems limp and useless. There is no suspicion of muscular rigidity or localized paralysis.

There were 13 of these unfortunate children, 5 boys and 8 girls, and the ages were from one to eleven. Nine appear to have been congenital, and in two the notes are indefinite. The labor was normal and easy on the testimony of the mother in 7 of the 9 cases, and the other two were premature deliveries. It was

very hard to find any assignable cause for these cases, the family history was good in 5 cases, twice phthisis was present, and in one case the mother had an anencephalic fœtus born previously; one attributed it to a fright in pregnancy, and the tendency is to assign it to any cause rather than inheritance. Dr. Down says of this tendency, "Parents always prefer to refer the cause to a post-uterine or non-congenital origin, partly because they think it frees them from a suspicion of hereditary influence, and partly from a notion that the child is more likely to be restored to its pristine state," and so this tendency of referring it to frights, etc., in pregnancy, arises.

Mitchell[30] tabulated the cases of 443 idiots that he saw consecutively, with regard to this, and found only 28 cases where there was any likelihood of its having been due to a fright or strong mental emotion during pregnancy, and in general he found this to be his experience in the examination of some 1500 idiots and imbeciles. As to convulsions, these were present in four cases, having begun before the child was a year old, and absent in five.

In the matter of intelligence, 11 of the 13 cases were low grade idiots, one could understand what was said to him, and one began at the age of four and a half to talk very imperfectly. Three could walk very unsteadily and one with the aid of a chair. All but one of the children had strabismus, and he was a child five years old with a head measuring 22 inches, which corresponds to a No. 7 hat, and large heads in this class were not uncommon; two children two years old, had heads of 19 and 20 inches, and one four years old measured 21 inches. This, of course, points to hydrocephalus as a common cause. The patellar reflexes were sometimes normal and sometimes increased,

[30] Obstet. Transactions, London, Vol. xxvi.

the legs were flabby, cool, and the hands and feet apt to be undeveloped. Disturbances of sensation were common, and one or two paid no attention when a pin was thrust into the leg. There was absence of the uvula in one of these cases also.

To enter upon the pathology of these cases would be to introduce the very extensive subject of the pathology of idiocy. Suffice it to say that all sorts of malformations of the skull and brain have been recorded, hypertrophy and atrophy [31] of the brain tissue have been described,[32] localized and disseminated sclerosis,[33] chronic hydrocephalus [34] and pressure from meningitis [35] are among the others.

Unlike the cases of spastic paralysis these children do not seem to have shown much of any tendency to improve in the time that they have been under observation, and of course any treatment further than mental training offers but little hope.

There are five miscellaneous cases which are unclassified. Two backward and rather feeble minded boys four and seven years old respectively, whose only disturbance is a tendency to hold the head over the right shoulder. There is beginning torticollis in one and no muscular alteration in the other; one was born by a forceps and the other by a natural labor. Both talk thickly and imperfectly. Little [36] called attention to the connection of cerebral injury and wry-neck.

The third was a girl who had slightly increased reflex excitability of the legs and a tendency to fall, the result, apparently, of long-continued epilepsy.

The other two were children who were very back-

[31] Cotard. There de Paris, 1868. Seibert. Arch f. Pediatrics, March, 1888, 168.
[32] Beach. Am. Jour. Ment. Sci., June, 1883, and April, 1881.
[33] Bunhuer. Arch. f. Psych., xii, 3.
[34] Tambarini. Revist Sperim, vi, 285.
[35] Seibert. Loc. cit.
[36] Little. Obstet. Trans., London, 1862.

ward about walking, but who ultimately recovered the full use of their limbs, and as they were in both cases suffering from rachitis, their disability was probably due to the condition known as rachitic paralysis, and, as they were originally considered cases of cerebral origin they have been included here.

As this paper is only a résumé, at best, no summary need be given more than to call attention to two or three practical points that seem to be evident. First, the great gravity of the prognosis of hemiplegia in childhood as regards mental enfeeblement, epilepsy, and possibly the transition to spastic paralysis. Again, that the influence of the difficulty of labor in the production of these cases has been very much overestimated. That the tendency of cases of spastic hemiplegia is to improve, with or without treatment. And lastly, that the existence of primarily spinal spastic paralysis in the child has yet to be proved anatomically, as well as the existence of the poliencephalitis of Strümpell.

NOTE.— Further bibliography can be found in the papers of McNutt, Am. Journ. Med. Sciences, January, 1885. Knapp, Journ. of Nervous and Mental Disease. Wallenburg, Jahrbuch f. Klide, 1885.

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